What's Hiding Under the Sink:

Dangers of Household Pesticides

Olivia James

In the war against home and garden pests, over 70 million American households make more than 4 billion pesticide applications per year. Indeed, 85% of America's 84.5 million households maintain a home arsenal averaging three to four pesticide products, ranging from pest strips, bait boxes, and bug bombs to flea collars, pesticidal pet shampoos, aerosols, granules, liquids, and dusts. There are over 20,000 different household pesticide products containing over 300 active ingredients and perhaps as many as 1,700 inert ingredients, according to the *National Home and Garden Pesticide Use Survey*, which was prepared for the EPA by the Research Triangle Institute in 1990. Seventy-five percent of American households use insecticides, with cockroaches and ants the leading targets.

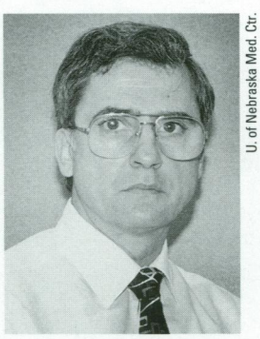
It's not just a disgust for bugs that prompts such widespread use. According to Tim Maniscalo, a company spokesperson for DowElanco, a manufacturer of chlorpyrifos, a leading pesticide ingredient, about 40% of the population is allergic to shed cockroach parts. Stings and bites from venomous pests such as fire ants and brown recluse spiders can be life threatening. Fleas, ticks, and mosquitoes are potential vectors of a wide range of diseases, ranging from bubonic plague to lyme disease to malaria. Thus, there are strong reasons for having household pesticide products available. Still, the pervasiveness of household pesticides makes the potential acute and chronic health effects of these products a matter of practical concern.

Nationwide in 1993, 140,000 pesticide exposures, 93% of which involved home use, were reported to poison control centers. About 25% of these reported exposures involved pesticide poisoning symptoms. Over half the exposures involved children under age 6, according to EPA health statistician and incident data officer Jerome Blondell. But Blondell worries that acute pesticide poisonings and poison control center statistics may be only the tip of the iceberg when it comes to the impact of household pesticide use on human health. Blondell says we could be misdiagnosing or overlooking chronic effects from some of today's common household pesticide products.

Carbamate Insecticides

Carbaryl and propoxur, carbamate insecticides introduced in 1956 and 1963, respectively, like most widely used household pesticides, have relatively low acute mammalian toxicities. "However," asks George Casale, research assistant professor at the University of Nebraska Medical Center's Eppley Research Institute, "are you deceiving yourself to think that because there is not acute exposure toxicity that you are safe?"

"The very safety of some of these pesticides could spell rather significant danger, because you can be exposed to quite a lot of pesticide with no concern," says Casale, who studies the immunological effects of common anticholinesterase pesticides, ranging from organophosphates like dichlorvos to carbamates like carbaryl. "People are too comfortable with some of these low acute toxicity pesticides, and they shouldn't be."


U. of Nebraska Med. Ctr.

George Casale—Lack of acute effects may not mean a pesticide is safe.

Anticholinesterase pesticides inhibit breakdown of the neurotransmitter acetylcholine by inhibiting acetylcholinesterase, a serine hydrolase enzyme. The possibility that carbaryl and other anticholinesterase insecticides affect more than the nervous system, for example, also impairing immune processes dependent on serine hydrolase activity, is usually ignored. However, in a series of experiments over the last several years, Casale and co-workers demonstrated that carbaryl and other common anticholinesterase insecticides inhibit serine hydrolase-dependent immune processes, such as interleukin 2 (IL-2) signaling.

Concentrations of carbaryl below those causing acute toxicity inhibit human natural killer cells *in vitro*. Natural killer cells are particularly effective against leukemias and lymphomas, which epidemiological studies have correlated with farm use of anticholinesterase pesticides around the world. In Casale's high-dose pilot study of mice, carbaryl, which is metabolized similarly in mice, rats, and humans, inhibited natural killer cells. Whole-animal studies of carbaryl and other anticholinesterase pesticides to determine whether natural killer cell inhibition occurs at low doses from repeated exposures, as would be the case in chronic household pesticide use, have yet to be concluded.

The next logical research step, says Casale, is to develop a whole-organism model to determine where at the cellular level the pesticide is affecting the immune system. Neither the kind of esterases inhibited by carbaryl and other anticholinesterase pesticides nor the actual esterase targets have been identified. Scientists do not know why carbaryl, despite its exceptionally low acute toxicity to the nervous system, is more toxic to the complement system than paraokon, the pri-

mary metabolite of the more acutely toxic pesticide parathion. "There has been very little in the way of a systematic approach to studying biological interactions with these chemicals," says Casale. Even in regard to cancer, it is hard to come up with general conclusions about these pesticides. "The support has been helter-skelter, not systematic."

"The problem that I have with transient acute effects," says Casale, "is that people are not exposed once to a chemical, but rather are exposed repeatedly." Even pesticides with very low acute toxicities can be so highly reactive with body proteins that crude pilot tests show 100% bonding with proteins within 24 hours. "There are probably quite a few chemicals out there, that . . . will modify proteins that the body will then recognize as foreign," he says. Casale decries the paucity of immunologic research on the many pesticides causing dermatitis and rashes, as these are likely candidates in processes related to allergy and autoimmune reactions.

According to Rudy Richardson, director of toxicology at the University of Michigan, much of the work on the immunologic effects of pesticides is difficult to interpret and equivocal, and there is not much in the way of controlled studies in humans. Richardson is hopeful that more immunologists will go into toxicology in the future. "We have spent an enormous amount of time in pesticides with cancer assessments," says John Bucher, acting chief of the toxicology branch of the Environmental Toxicology Program at NIEHS. "[But] we could be missing the boat on the potential effects on the immune system. What we see is an increasing number of reports on multiple chemical sensitivity, which anecdotally has been set off in people by one large exposure to a pesticide or multiple pesticides." Bucher believes that there is some immune system involvement in multiple chemical sensitivity and that the role of pesticides needs more study. Also insufficiently studied are subtle nervous system effects from pesticide exposures. "We almost never see anything on learning, memory, and potential psychological effects of exposures," adds Bucher. "You can't ask a test animal for the kind of information that you can ask people. So you can't adequately study some of these things with animal models."

Organophosphate Insecticides

Chlorpyrifos, an anticholinesterase organophosphate that is among the 10 most commonly used household insecticides, has been in use since 1966. Neonatal animals generally show a higher sensitivity to organophosphate insecticides than older animals, and chlorpyrifos is no exception. Human newborns have very low concentrations of the serum enzyme needed to detoxify chlorpyrifos, says Clement Furlong, director of the toxicology program at the University of Washington. Furlong and graduate student Wan-Fen Li found that newborn rodents require several weeks to develop the enzymes needed to detoxify chlorpyrifos. Furlong is currently studying how long it takes human newborns to develop the serum enzyme needed for chlorpyrifos detoxification.

NCI

Sheila Zahm—The whole idea is prudent avoidance to minimize pesticide exposure.

Detoxification of organophosphate pesticides is genetically controlled in humans and other species, and at least 15-fold differences exist among humans in their ability to hydrolyze the toxic metabolite of chlorpyrifos. This biochemical individuality may help explain the variation in symptoms from similar pesticide exposures. When Furlong studied New York City pesticide applicators applying chlorpyrifos full-time, all had the resistant phenotype. Others are less fortunate in their genetic inheritance. Permanent cognitive damage manifest as a substantial drop in IQ to below normal was the outcome for a physician accidentally poisoned by chlorpyrifos. In other cases, including many instances of neurodegenerative disorders liked delayed neuropathy, the damage is more transient, and the person eventually recovers.

"I have some concern regarding the over-the-counter sales of these compounds because the average householder does not have a clue how damaging these pesticides can be if misused," says Furlong, who half-jokingly concludes seminars by telling those with low levels of detoxification enzyme to switch to fly swatters. Actually, the genetics of detoxification are quite complicated: a two-step activation and breakdown pathway is controlled by different genes. Also, genetic resistance to one pesticide does not necessarily mean resistance to others. For example, people with the genotype most resistant to parathion are most susceptible to diazinon and vice versa. There are also environmental influences, such as smoking and drugs, which may increase sensitivity by speeding up P450 microsome activation of chlorpyrifos into its neurotoxic oxon metabolite.

"One possibility with an insecticide such as chlorpyrifos that can cause extensive neurochemical changes in the absence of overt signs is that significant exposures can occur with less indication of exposure," says Carey Pope, director of the toxicology program at Northeast Louisiana University. Long-term neurochemical and behavioral effects of chlorpyrifos on the brain may be cryptic, easily overlooked, and persist in adults after a single exposure without overt signs of toxicity.

"The adult brain appears more sensitive to persistent neurochemical changes, compared to the neonatal brain," says Pope, citing chlorpyrifos experiments using the maximum tolerated dose, the highest dose of a chemical causing no lethality. About 50% of the maximum tolerated dose of chlorpyrifos inhibits brain neurochemicals in young animals, whereas about 15% of the maximum tolerated dose causes a similar 50% inhibition of acetylcholinesterase activity in adult brains. Richardson, who recently reviewed the literature on the neurotoxic potential of chlorpyrifos in the *Journal of Toxicology and Environmental Health*, emphasizes that chlorpyrifos exhibits only moderate acute toxicity in most mammalian species because the active oxon metabolite is detoxified. When problems like delayed neurotoxicity occur, it is usually associated with extremely large doses of the insecticide, well above those encountered in normal household use.

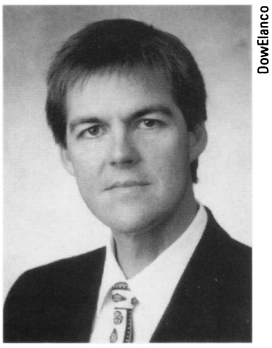
Another organophosphate insecticide with possible brain effects, dichlorvos, is found in 8.3 million households and applied

Top Ten Home and Garden Pesticides

Active ingredient	Thousands products	Percentage products	Thousands households	Percentage households	Thousands applications indoors	Thousands applications outdoors
Piperonyl butoxide (synergist)	41,729	12.76	27,335	34.01	294,013	58,991
Pyrethrins	34,609	10.58	22,739	28.46	244,328	39,289
MGK-264) (synergist)	27,558	8.43	19,532	24.51	203,328	13,249
Propoxur	21,484	6.57	18,749	23.71	209,528	53,594
DEET	21,544	6.59	17,227	21.78	238,433	14,134
Aliphatic petroleum hydrocarbons	18,652	5.70	14,480	18.27	110,701	32,750
Carbaryl	18,437	5.64	12,494	15.77	28,591	31,735
Phenylphenol	17,618	5.39	16,227	20.63	537,048	1,452
Bleach	16,266	4.97	15,591	19.95	672,959	10,397
Chlorpyrifos	16,652	5.09	13,993	17.81	174,322	41,900

Source: U. S. Department of Commerce

72 million times per year, mostly indoors via foggers and pest strips. "Indoor air use of pesticide products in the home is the main source of exposure for children," says toxicologist William Pease of the University of California-Berkeley School of Public Health. Pease asserts that exposures from household use exceed those from pesticide residues on food. His review of San Francisco Bay area poison control center records revealed cases of children becoming sick after crawling on freshly sprayed floors and carpet. "Basically, pesticide products do not need active ingredient coating all spaces in the home," says Pease.



DowElanco

Eric Wintemute—Industry has made great improvements toward safer products.

Pease also expresses concern about *para*-dichlorobenzene moth balls and other products designed for continuous pesticide release in closed spaces, where concentrations may become elevated. Pease cites regulatory EPA calculations that a child who is active during 6 out of 24 hours of confinement in a room treated with a dichlorvos fogger can absorb a dose exceeding that causing cholinesterase inhibition in chronic animal experiments, resulting in symptoms such as runny eyes, diarrhea, or nausea.

Though dichlorvos is hardly unique in its anticholinesterase mode of action, it has been unfairly singled out by the media and antipesticide groups for criticism on the basis of health effects studies that have been misinterpreted and even later been shown to be wrong, says Eric Wintemute, president and CEO of Amvac Chemical Corporation, which makes dichlorvos. "Industry as a whole has made great improvements towards more environmentally friendly products," says Wintemute.

Dichlorvos and other organophosphates replaced the older, more persistent organochlorine insecticides like DDT because they break down relatively quickly and do not have long-lasting residues. "As far as household pest control, we certainly agree that the first line of defense should be screens and mechanical control of flying pests," says Wintemute. Baits and traps, which are alternatives to spraying a whole area with pesticides, are becoming more popular and work well against crawling insects like roaches, but are not so effective for flying insects, he says. "At the point where pests become a nuisance, because of the health risk, as insects do spread disease, this is when to consider pesticides," says Wintemute. "Unlike aerosols, pest strips are controlled release to decrease levels of the target pest. Pest strips can be formulated so that there is a lower level of exposure than with aerosols, so there is less possibility of misuse."

EPA

Louise Mehler—There are inert ingredients of real toxicological significance.

Use of dichlorvos for controlled release from pet flea collars was stopped in the mid-1980s, following reaction to a "Today Show" vignette on network television that showed a child petting a cat while a voice cited a study saying that children had a 100 times greater chance of getting cancer when the pet had a flea collar containing dichlorvos. "It was emotional, an overreaction, fear-driven," said Wintemute, in describing how Hartz, and then other pet flea collar companies, stopped using dichlorvos and switched to competing active ingredients, such as chlorpyrifos.

The positive cancer study in question was conducted by the National Toxicology Program (NTP) and conflicted with 10 published studies that were negative. According to Wintemute, a review by the EPA Science Advisory Panel found serious problems with the NTP study and interpretation of the data. For instance, there was no allowance for the fact that rat tumors in the study were benign, not malignant, or that, unlike mice, humans do not have a forestomach subject to feeding tube irritation, or that doses were relatively high and the aging rats were tumor-prone. But the political climate in 1987 was such that the EPA reclassified dichlorvos as a probable carcinogen on the basis of one partially positive and 10 negative studies, said Wintemute. In 1989, after new studies and a special review, the EPA reclassified dichlorvos from a probable carcinogen to a possible carcinogen. "We are working to move dichlorvos to a group d classification [insufficient evidence to determine potential carcinogenicity] or group e [not a proven carcinogen], where we feel it belongs," says Wintemute.

In 1992, Japan completed its review of dichlorvos and concluded that it was not a human carcinogen. In 1993, the World Health Organization concluded that dichlorvos was not a chronic health hazard. In 1994, The United Kingdom concluded that no classification was required for human carcinogenicity and that dichlorvos was not a mutagen. Yet dichlorvos on pest strips was associated with childhood leukemia in a recent epidemiological case–control study in Denver, Colorado, published in the *American Journal of Public Health*. However, Wintemute questions the scientific value of answers obtained when parents of leukemia victims are asked if they had used pest strips.

In a separate case–control study in Missouri by James Davis, an epidemiologist with the Missouri Department of Health, dichlorvos and other insecticides were associated with an elevated odds ratio for childhood brain cancer. "The true extent of exposure and health problems associated with consumer pesticide use are currently unknown," says Davis, noting that the epidemiological studies "raise some red flags that should be looked at," particularly since no primary cause has been identified for childhood brain cancer.

"If epidemiological studies are detailed enough to indicate certain agents and correlate with laboratory studies, that can certainly be very powerful evidence," says Sheila Zahm of the National Cancer Institute, referring to Davis's studies. However, Zahm is also quick to add that epidemiological leads are not always confirmed by animal studies. A major case in point is the widely used lawn herbicide 2,4-D, which is found in 10.5 million households. Epidemiological studies with farmers linked 2,4-D use with lymphomas. However, animal studies later vindicated 2,4-D as not being a carcinogen.

There are many limitations and variables which must be kept in mind when evaluating epidemiological studies, says Richardson. Results can be influenced by how questions are asked, and human memory can be selective. People may remember a pesticide spray but forget dietary or other potential causal factors, or vice versa. Also, epidemiological studies typically lack exposure data at the part-per-million level and lack blood cholinesterase measurements that would be of most value to toxicologists. "We need more basic toxicology and more controlled studies," says Richardson. "If we had double-blind prospective studies, instead of retrospective studies, then we could draw some real conclusions."

In a recent Norwegian study published in *Neurochemical Research* in 1994, researchers fed dichlorvos to pregnant guinea pigs and found a dose-dependent reduction in brain weights of offspring unrelated to either body weight changes or specific neurotransmitters or brain regions monitored. Dichlorvos alkylation of neuronal DNA early in development, such as during the brain growth spurt period (days 40–50 of gestation in guinea pigs) before DNA repair enzymes become active, is suspected.

The best way to deal with fetal and infant pesticide sensitivity, contends

Wintemute, is to have a 100-fold buffer zone above the no-effect level so that adverse effects are unlikely even if the product is abused. Though protocols exist for exposing pregnant animals to a chemical and examining the second generation for mutagenesis and teratogenicity, says Zahm, "there is nothing specific if a pesticide is a suspected child carcinogen. There are standard testing protocols, but often the target sites in animals are not the same as in humans." So it would be difficult, barring breakthroughs in testing protocols, to check, for instance, for brain cancer risk in children using current standard animal testing protocols. "The whole idea is prudent avoidance to minimize exposure, especially to children, and certainly if pregnant to try not to use anything," says Zahm, noting that rapidly growing fetuses may be more susceptible to mutagenesis, chromosomal aberrations, and carcinogenesis. Zahm also points out that infants crawling around on carpets can be affected by lawn and other outdoor pesticides tracked indoors. When these chemicals are brought indoors, the residues last much longer than outdoors, where water and sunlight promote biodegradation. Both Zahm and Davis believe that more studies need to focus on the fetus and on infants from birth to age 6 months, as these are critical periods of susceptibility.

Petroleum Hydrocarbons

Aliphatic petroleum hydrocarbons are the sixth most common active ingredient in household pesticides today. Highly refined horticultural oil has a relatively low acute oral toxicity, though it is a skin and lung irritant. Petroleum oils vary greatly in terms of refining, and hence in amounts of aromatic hydrocarbon impurities, which are potentially toxic benzene-ring compounds. Thus, petroleum oil-based pesticides are complex mixtures of varying quantities of aromatic and aliphatic hydrocarbons with potentially diverse toxicological profiles and health effects.

Among the few toxicological effects of aliphatic petroleum hydrocarbons mentioned in a 1988 petroleum industry review in *Occupational Medicine* are central nervous system depression manifested as dizziness and incoordination. Petroleum hydrocarbons and solvents of various sorts, including aromatic compounds such as benzene, toluene, and xylene, are also among the unnamed "inert ingredients" formulated into household pesticide products.

Synergists and Pyrethrins

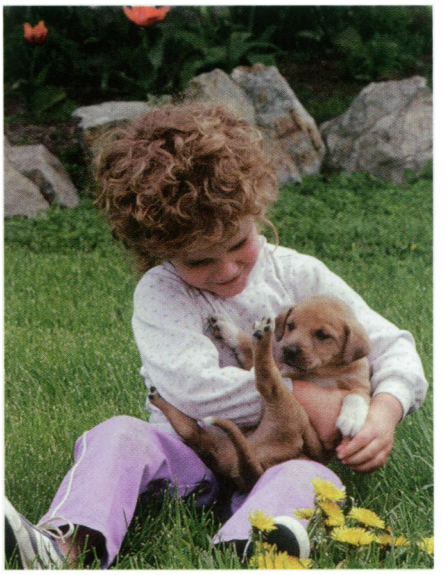
The synergist piperonyl butoxide (PBO), the number one active ingredient in household pesticides, is commonly formulated with the number two active ingredient, pyrethrin compounds, in household pesticide products. Synergists by themselves have little pesticidal activity, but increase the effectiveness of other pesticide active ingredients. PBO has an extremely low acute oral toxicity. By itself, PBO has, at least until recently, been considered neither mutagenic nor carcinogenic, though liver and kidney damage has been noted over the years in animal studies. However, Japanese researchers at the Tokyo Metropolitan Laboratory of Public Health have recently published a series of chronic toxicity studies that shows a dose-dependent relationship between hepatocellular carcinoma and PBO when doses are increased to exceptionally high levels, well above what human beings are ever likely to encounter.

Ironically, if a synergist like PBO were banned as a carcinogen, higher amounts of other pesticide active ingredients would be added to the environment because synergists allow dramatic reductions in quantities of active ingredients needed for the pesticide to be effective.

The third most common active ingredient is the synergist MGK-264, which is applied over 200 million times per year. The *Hazardous Chemicals Desk Reference* refers to MGK-264 as being of moderate toxicity, with central nervous system and reproductive effects in experimental animals. Surprisingly, for such a widely used household pesticide ingredient, MGK-264 is not currently the subject of much toxicological research.

Pyrethrins are the collective name for a group of six pesticidal compounds derived from pyrethrum flowers in the genus *Chrysanthemum*. Pyrethrum flowers and refined pyrethrin extracts with varying amounts of floral impurities, some of which are allergens, have been used in pest control for several centuries. Though pyrethrum extracts are relatively low in terms of acute toxicity, there is concern that pyrethrins and their synthetic counterparts, pyrethroids, can trigger allergic reactions, particularly among the nation's estimated 15 million persons with asthma.

A 1994 report by Paul Wax, a physician at the Strong Memorial Hospital in Rochester, New York, published in *Clinical Toxicology,* reported the death of a 37-year-old woman with a history of mild asthma after inhaling a pyrethrin pet shampoo. Minutes after applying the shampoo, the woman developed fatal lung symptoms, went into cardiopulmonary arrest, and died. However, said Wax, the 0.06% pyrethrins in the pet flea shampoo were not proven to be the cause, as there were neither immunological studies of the event nor subsequent animal studies trying to reproduce the result. The report was strictly observational, as is often the case in pesticide exposure incidents. Still, after the shampoo ingredients and emulsifiers listed on the label were excluded as allergens, pyrethrins were the only known allergen the woman could have been exposed to. However, 54% of the flea shampoo was labeled inert ingredients, which are considered trade secrets not divulged even to the medical profession.



At play among pesticides. Infants and children are at greater risk from exposure to pesticides on lawns and pets.

Several derivatives of natural pyrethrin molecules, known as synthetic pyrethroids, are also widely used household pesticides and are suspected to be allergens. Some of these include tetramethrin, resmethrin, and allethrin, cumulatively found in over 30 million households. Thus, pyrethrin and pyrethroid products may need to be labeled with bronchospasm warnings for asthmatics.

Inert Ingredients

The EPA estimates that there are at least 1,700 chemical compounds collectively listed under the rubric "inert ingredients" on pesticide labels. A recent walk down a supermarket insecticide aisle revealed many products labeled as over 99% inert ingredients. Section 2m of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) states: "The term 'inert ingredient' means an ingredient which is not active." In actual practice, pesticide manufacturers decide what to call inert and what to designate as an active ingredient subject to EPA regulation. This has produced a situation where ingredients considered active and regulated by the EPA in some pesticide products are unregulated, inert ingredients missing from the labels of other pesticide products. The EPA has produced several categories of inert ingredients, which include several of the top household pesticide active ingredients. According to the EPA's Office of the Inspector General, "EPA knows little or nothing about the adverse effects of most of

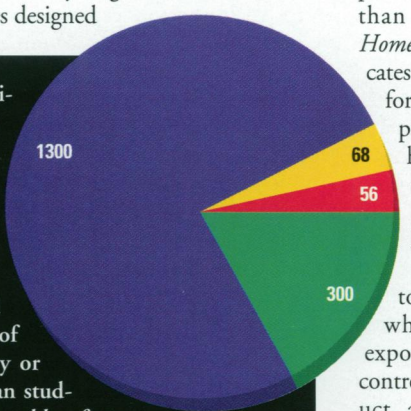
these inerts [inerts of unknown toxicity]. Some data may exist for the inerts of unknown toxicity, but EPA has not yet evaluated the data to determine the effects."

Inert ingredients are low priority, accounting for under 1% of the Office of Pesticide Programs budget, as the EPA still has many older (pre-1972) active ingredients that need to be reregistered and evaluated for health effects under FIFRA. Also, EPA has no specific procedures or timeframes for ensuring that these inerts are reviewed, according to the EPA's Office of the Inspector General. "Until these reviews are completed, users are unaware of potentially toxic inert ingredients contained in certain pesticide products. The use of these pesticide products may be jeopardizing human health and the environment," states the office.

"Inert ingredients are confidential information," adds California-EPA's Louise Mehler, a physician and program director of CAL-EPA's Worker Pesticide Illness Surveillance Program. "If we were to disclose that information we could be prosecuted for it and imprisoned. There are inert ingredients that are sometimes of real toxicological significance. It could also be just water." Though inerts are trade secrets protected by law from disclosure, it is widely believed that pesticide companies know their competitors' inert ingredients, as reverse engineering is relatively simple with today's technology. "The chemists here say that since the invention of the mass spectrometer anybody who wants can really find out," says Mehler.

Not all chemical companies are rigorously secretive, and some reveal their inert ingredients upon request. For example, DowElanco makes no secret in its technical literature that its liquid formulations of chlorpyrifos are "usually solutions of chlorpyrifos in a petroleum fraction" referred to as "xylene range aromatic solvent." The major components of this solvent are nine-carbon aromatic hydrocarbons with some xylene. Xylene is sometimes registered as an active ingredient, as it is has pesticidal activity, but it is more commonly used as an inert ingredient to keep the pesticide active ingredient in solution, prevent clumping, and as a delivery vehicle.

Xylene, toluene, and ethyl benzene are among the inerts found in common household products studied by John Wurpel, associate professor at St. John's University College of Pharmacy. Potential health effects of these inert ingredients include nonspecific depression of the central nervous system. Benzene is a known carcinogen. Xylene, at least in the case of chlorpyrifos, also has synergistic health effects. In rat studies designed to examine behavioral effects of low pesticide doses similar to those found in homes, "We were surprised to see birth effects [embryotoxicity] because we used a low dose," said Wurpel. Usually chlorpyrifos is a teratogen only at high doses. But in combination with xylene there is a synergism, probably because the xylene carrier allows the pesticide to enter the fetus. Thus, inert ingredients in household pesticide formulations can complicate interpretation of health effects based on pure active ingredients.



Potentially toxic—Inert ingredients with a high priority for testing. Many of these are structurally similar to other chemicals that exhibit toxicity. Either testing is already underway for these potentially toxic ingredients, or the existing data suggests potential adverse effects. *Examples: toluene, xylene, petroleum hydrocarbons, methyl bromide*

Toxic—Inert ingredients with known adverse effects and of toxicological concern. These ingredients have evidence of carcinogenicity, adverse reproductive effects, neurotoxicity or other chronic effects, or birth defects in laboratory or human studies. *Examples: aniline, asbestos, benzene, carbon disulfide, chloroform, formaldehyde; hexachlorophene, lead, cadmium, mercury oleate, pyrethrins, and pyrethroids*

Generally recognized as safe—Inert ingredients for which EPA has no reason to expect adverse effects to occur. These include ingredients of minimal concern and for which sufficient information is available to conclude that adverse effects are not expected. *Examples: alfalfa, cardboard, castor oil, dextrose, ethanol, fish meal, gypsum, lard, latex, nylon, olive oil, onions, pine oil, polyvinyl chloride resin, rubber, silicone, sodium fluoride, urea, water, wintergreen oil*

Unknown toxicity—EPA knows little or nothing about the adverse effects of most of these inert ingredients . Some data may exist for the inert ingredients of unknown toxicity, but EPA has not yet evaluated the data to determine the effects. These ingredients presented no cause for suspicion. An inert ingredient was put in this group if there was no basis to put it in any of the other three groups. *Examples: barium sulfate, epoxy resin, aluminum powder, styrene acrylic copolymer, sodium nitrite, sulfuric acid, salicylic acid, limonene, thymol, menthol, lithium chloride, napthalene, polyethylene terephthalate, D and C Red # 37, saccharin, malathion, kerosene, coal tar, asphalt, lanolin, camphor, boric acid, Freon 114*]

Source: U.S. EPA Office of the Inspector General, *Inert Ingredients of Pesticides* (audit report no. E1EPF1-05-0117-1100378), 27 September 1991.

Future Directions

A 1994 study of pesticide labels published in the *Journal of the American Optometric Association* found that it requires an 11th-grade cognitive reading level to understand a pesticide label, which means that 40–50% of the general population cannot read and understand the directions on a pesticide product label, assuming they have the necessary 20/30 visual acuity to read the fine print. This study suggests that labeling may not be even minimally effective in protecting the common user of household pesticides from adverse health effects.

In addition, some experts suspect that there may be a lot of avoidable urban pesticide exposures because people may be using pesticides out of annoyance or fear, rather than actual need. Indeed, the *National Home and Garden Pesticide Use Survey* indicates that 37% of all U.S. households treat for insects even when there is not a major problem. However, almost 39% of households use insecticides because they have a major insect problem, often of pests of potential public health importance.

There is a trend toward use of less toxic alternatives, like baits and traps, which minimize household pesticide exposures. "Because of the difficulties in controlling how the end-user uses the product, and knowing that at least some will become ill, as we are currently seeing adverse effects, the question in our minds, since there are alternative means of treating many pests, is if we should even recommend some of these products when we know that there are alternatives," says Pease. Experts agree, however, that it would be premature to call a truce against pests and jettison household pesticides, despite potential health risks, until we have effective alternatives available. Until that time, efforts in the war against bugs should include systematic research into the potential chronic health effects of the most widely used household pesticides and their potential replacements.

Joel Grossman

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